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NEWS 6	FEB 10	COMPENDEX reloaded and enhanced
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NEWS 13	FEB 23	Three million new patent records blast AEROSPACE into STN patent clusters
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NEWS 15	MAR 06	INPADOCDB and INPAFAMDB enhanced with new display formats
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NEWS 17	MAR 11	ESBIOBASE reloaded and enhanced
NEWS 18	MAR 20	CAS databases on STN enhanced with new super role for nanomaterial substances
NEWS 19	MAR 23	CA/CAplus enhanced with more than 250,000 patent equivalents from China
NEWS 20	MAR 30	IMSPATENTS reloaded and enhanced
NEWS 21	APR 03	CAS coverage of exemplified prophetic substances enhanced
NEWS 22	APR 07	STN is raising the limits on saved answers
NEWS 23	APR 24	CA/CAplus now has more comprehensive patent assignee information
NEWS 24	APR 26	USPATFULL and USPAT2 enhanced with patent assignment/reassignment information
NEWS 25	APR 28	CAS patent authority coverage expanded
NEWS 26	APR 28	ENCOMPLIT/ENCOMPLIT2 search fields enhanced
NEWS 27	APR 28	Limits doubled for structure searching in CAS REGISTRY
NEWS 28	MAY 08	STN Express, Version 8.4, now available
NEWS 29	MAY 11	STN on the Web enhanced

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 NEWS 30  MAY 11  BEILSTEIN substance information now available on
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 NEWS 31  MAY 14  DGENE, PCTGEN and USGENE enhanced with increased
                  limits for exact sequence match searches and
                  introduction of free HIT display format
 NEWS 32  MAY 15  INPADOCDB and INPAFAMDB enhanced with Chinese legal
                  status data
 NEWS 33  MAY 28  CAS databases on STN enhanced with NANO super role in
                  records back to 1992

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=> S 110117-83-4/RN
L1            1 110117-83-4/RN
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=> DIS L1 1 SQIDE

L1   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2009 ACS on STN
RN   110117-83-4  REGISTRY
CN   D-Tryptophan, 1-methyl-  (CA INDEX NAME)
OTHER NAMES:
CN   2: PN: WO2007050405 PAGE: 28 claimed sequence
CN   D-(+)-1-Methyltryptophan
CN   D-1-Methyltryptophan
FS   STEREOSEARCH
MF   C12 H14 N2 O2
CI   COM
SR   CA
LC   STN Files:   AGRICOLA, BEILSTEIN*, CA, CAPLUS, CASREACT, CHEMCATS,
       PROUSDDR, TOXCENTER, USPAT2, USPATFULL
         (*File contains numerically searchable property data)
DT.CA  CAplus document type:  Journal; Patent
RL.P   Roles from patents:  BIOL (Biological study); RACT (Reactant or
       reagent); USES (Uses)
RL.NP  Roles from non-patents:  ANST (Analytical study); BIOL (Biological
       study); PREP (Preparation); PROC (Process); PRP (Properties); RACT
       (Reactant or reagent); USES (Uses)

Absolute stereochemistry.
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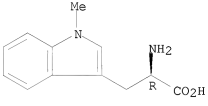


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              35 REFERENCES IN FILE CA (1907 TO DATE)
              35 REFERENCES IN FILE CAPLUS (1907 TO DATE)


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                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       2.53       2.75

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FILE COVERS 1907 - 29 May 2009  VOL 150 ISS 23
FILE LAST UPDATED: 28 May 2009  (20090528/ED)
REVISED CLASS FIELDS (/NCL) LAST RELOADED:  Feb 2009
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE:  Feb 2009

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reclassification data for the third quarter of 2008.

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=> s l1
L2          35 L1

=> s l2 and (?cancer? or ?tumor? or ?tumour? or ?neoplasm?)
        455592 ?CANCER?
        724068 ?TUMOR?
          6288 ?TUMOUR?
          6288 ?TUMOUR?
        724432 ?TUMOR?
                 (?TUMOR? OR ?TUMOUR?)
          6288 ?TUMOUR?
        724068 ?TUMOR?
        724068 ?TUMOR?
        724432 ?TUMOUR?
                 (?TUMOUR? OR ?TUMOR?)
        562688 ?NEOPLASM?
L3          13 L2 AND (?CANCER? OR ?TUMOR? OR ?TUMOUR? OR ?NEOPLASM?)

=> d l3 1-13 ibib, abs

L3   ANSWER 1 OF 13  CAPLUS  COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER:         2008:1289170  CAPLUS
DOCUMENT NUMBER:          150:443513
TITLE:                    IDO1 and IDO2 are expressed in human tumors:
                          levo- but not dextro-1-methyl tryptophan inhibits
                          tryptophan catabolism
AUTHOR(S):                Loeb, Stefan; Koenigsrainer, Alfred; Zieker, Derek;
                          Bruecher, Bjoern L. D. M.; Rammensee, Hans-Georg;
                          Opelz, Gerhard; Terness, Peter
CORPORATE SOURCE:         Department of General, Visceral and Transplant
                          Surgery, University Hospital of Tuebingen, Tuebingen,
                          72076, Germany
SOURCE:                   Cancer Immunology Immunotherapy (2009), 58(1), 153-157
                          CODEN: CIIMDN; ISSN: 0340-7004
PUBLISHER:                Springer
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
AB   Objectives Indoleamine-2,3-Dioxygenase (IDO) is an immunosuppressive mol.
     inducible in various cells.  In addition to classic IDO (IDO1), a new
     variant, IDO2, has recently been described.  When expressed in dendritic
     cells (DCs) or cancer cells, IDO was thought to suppress the
     immune response to tumors.  A novel therapeutic approach in
     cancer envisages inhibition of IDO with 1-methyl-tryptophan (1MT).
     The levo-isoform (l-1MT) blocks IDO1, whereas dextro-1MT (d-1MT), which is
     used in clin. trials, inhibits IDO2.  Here we analyze IDO2 expression in
     human cancer cells and the impact of both 1-MT isoforms on IDO
     activity.  Methods: Surgically extirpated human primary tumors
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as well as human cancer cell lines were tested for IDO1 and IDO2 expression by RT-PCR. IDO1 activity of Hela cells was blocked by transfection with IDO1-specific siRNA and analyzed for tryptophan degradation by RP-HPLC. The impact of d-1MT and l-1MT on IDO activity of Hela cells and protein isolates of human colon cancer were studied. Results: Human primary gastric, colon and renal cell carcinomas constitutively expressed both, IDO1 and IDO2 mRNA, whereas cancer cells lines had to be induced to by Interferon-gamma (IFN-γ). Treatment of Hela cells with IDO1-specific siRNA resulted in complete abrogation of tryptophan degradation Only l-1MT, and not d-1MT, was able to block IDO activity in IFN-γ-treated Hela cells as well as in protein isolates of primary human colon cancer. Conclusions: Although IDO2 is expressed in human tumors, tryptophan degradation is entirely provided by IDO1. Importantly, d-1MT does not inhibit the IDO activity of malignant cells. If ongoing clin. studies show a therapeutic effect of d-1MT, this cannot be attributed to inhibition of IDO in tumor cells.

REFERENCE COUNT: 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 2 OF 13 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2008:1034489 CAPLUS
DOCUMENT NUMBER: 149:486285
TITLE: Interaction of tryptophan derivatives with SLC6A14 (ATB0,+) reveals the potential of the transporter as a drug target for cancer chemotherapy
AUTHOR(S): Karunakaran, Senthil; Umapathy, Nagavedi S.; Thangaraju, Muthusamy; Hatanaka, Takahiro; Itagaki, Shiro; Munn, David H.; Prasad, Puttur D.; Ganapathy, Vadivel
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, Medical College of Georgia, Augusta, GA, 30912, USA
SOURCE: Biochemical Journal (2008), 414(3), 343-355 CODEN: BIJOAK; ISSN: 0264-6021
PUBLISHER: Portland Press Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB ATB0,+ [SLC6A14 (solute carrier family 6 member 14)] is an Na+/Cl--coupled amino acid transporter whose expression is up-regulated in cancer . 1-Methyltryptophan is an inducer of immune surveillance against tumor cells through its ability to inhibit indoleamine dioxygenase. In the present study, we investigated the role of ATB0,+ in the uptake of 1-methyltryptophan as a potential mechanism for entry of this putative anticancer drug into tumor cells. These studies show that 1-methyltryptophan is a transportable substrate for ATB0,+. The transport process is Na+/Cl--dependent with an Na+/Cl-/1-methyltryptophan stoichiometry of 2:1:1. Evaluation of other derivs. of tryptophan has led to identification of α-methyltryptophan as a blocker, not a transportable substrate, for ATB0,+. ATB0,+ can transport 18 of the 20 proteinogenic amino acids. α-Methyltryptophan blocks the transport function of ATB0,+ with an IC50 value of .apprx.250 μM under conditions simulating normal plasma concns. of all these 18 amino acids. These results suggest that α-methyltryptophan may induce amino acid deprivation in cells which depend on the transporter for their amino acid nutrition. Screening of several mammary epithelial cell lines shows that ATB0,+ is expressed robustly in some cancer cell lines, but not in all; in contrast, non-malignant cell lines do not express the transporter. Treatment of ATB0,+-pos. tumor cells with α-methyltryptophan leads to suppression of their colony-forming ability, whereas ATB0,+-neg. cell lines are not affected. The blockade of ATB0,+ in these cells with α-methyltryptophan is associated with cell cycle arrest. These studies

reveal the potential of ATB0,+ as a drug target for cancer chemotherapy.

REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 3 OF 13 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2008:1012413 CAPLUS
DOCUMENT NUMBER: 149:283064
TITLE: Chemotherapeutic targeting of indoleamine 2,3-dioxygenase, pd-1/pd-l pathways, and ctla4 pathways in the activation of regulatory t cells
INVENTOR(S): Sharma, Madhav D.; Blazar, Bruce R.; Mellor, Andrew L.; Munn, David H.
PATENT ASSIGNEE(S): Medical College of Georgia Research Institute, Inc., USA; Regents of the University of Minnesota
SOURCE: PCT Int. Appl., 108pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2008100562	A2	20080821	WO 2008-US1946	20080214
WO 2008100562	A3	20081120		

W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, NO, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

PRIORITY APPLN. INFO.: US 2007-901229P P 20070214
US 2007-959053P P 20070711

AB The present invention includes methods of enhancing immune responses by administering an inhibitor of indoleamine-2,3-dioxygenase (IDO) along with one or more inhibitors of the PD-1/PD-L pathway and/or one or more inhibitors of the CTLA4 pathway. Administration of IDO inhibitor 1-methyl-tryptophan combined with cyclophosphamide significantly reduced Treg suppressor activity in tumor draining lymph nodes.

L3 ANSWER 4 OF 13 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2008:421542 CAPLUS
DOCUMENT NUMBER: 149:227
TITLE: Differential targeting of tryptophan catabolism in tumors and in tumor-draining lymph nodes by stereoisomers of the IDO inhibitor 1-methyl-tryptophan
AUTHOR(S): Muller, Alexander J.; Metz, Richard; Prendergast, George C.
CORPORATE SOURCE: Lankenau Institute for Medical Research, Wynnewood, PA, USA
SOURCE: International Congress Series (2007), 1304(Interdisciplinary Conference on Tryptophan and Related Substances: Chemistry, Biology, and Medicine, 2006), 250-261

CODEN: EXMDA4; ISSN: 0531-5131
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Increased activity of the tryptophan-catabolizing enzyme indoleamine 2.3-dioxygenase (IDO), encoded by the INDO gene, has been associated with a broad spectrum of cancers and is implicated in the pathophysiol. process of tumoral immune escape. Our interest in IDO grew out of the finding that disruption of the Bin1 anti-cancer gene in oncogenically transformed mouse cells can lead to elevated interferon-γ mediated induction of Indo gene expression that is associated with immune escape. Using the prototypical IDO inhibitor 1-methyl-tryptophan (1MT), we demonstrated synergistic cooperativity with cytotoxic chemotherapy in an autochthonous mouse breast cancer model. Of the two stereoisomers of 1MT, the D isomer has been demonstrated to be a substantially less potent inhibitor of the IDO enzyme. However, in tolerogenic, IDO-expressing dendritic cells (DCs), D-1MT is as effective as L-1MT at blocking tryptophan catabolism and is actually superior at abrogating T cell suppression. This is consistent with data obtained in two mouse breast cancer models in which IDO is predominantly expressed in DCs within the tumor-draining lymph nodes. In both of these models D-1MT was more effective than L-1MT as an anti-tumor agent. We have recently discovered that a previously undocumented, IDO-related enzyme, referred to here as IDO2, is preferentially inhibited by D-1MT. The relative importance of targeting IDO vs. IDO2 with inhibitory compds. and the possibility of cross-talk between these two enzymes is currently being evaluated.
REFERENCE COUNT: 62 THERE ARE 62 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 5 OF 13 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2008:243141 CAPLUS
DOCUMENT NUMBER: 148:553032
TITLE: Levo- but not dextro-1-methyl tryptophan abrogates the IDO activity of human dendritic cells
AUTHOR(S): Lob, Stefan; Konigsrainer, Alfred; Schafer, Richard; Rammensee, Hans-Georg; Opelz, Gerhard; Terness, Peter
CORPORATE SOURCE: Department of General, Visceral and Transplant Surgery, University Hospital of Tubingen, Tubingen, Germany
SOURCE: Blood (2008), 111(4), 2152-2154
CODEN: BLOOAW; ISSN: 0006-4971
PUBLISHER: American Society of Hematology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Clin. trials were started with the aim of inducing tumor immunity by blocking the immunosuppressive action of indoleamine-2,3-dioxygenase (IDO) with the IDO2-inhibitor dextro-1-methyl-Trp (D-1MT). Here we show that human dendritic cells (DCs) express both IDO-1 and IDO-2, but that only IDO1 mediates tryptophan catabolism; furthermore, its activity is blocked by levo-1MT, whereas D-1MT is inefficient. Consequently, in humans any possible antitumor effects of D-1MT cannot be attributed to abrogation of IDO activity in DCs as described in this study.
REFERENCE COUNT: 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 6 OF 13 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2007:1443910 CAPLUS
DOCUMENT NUMBER: 148:440193
TITLE: Toxicology and pharmacokinetics of 1-methyl-D-tryptophan: Absence of toxicity due to

saturating absorption

AUTHOR(S): Jia, Lee; Schweikart, Karen; Tomaszewski, Joseph; Page, John G.; Noker, Patricia E.; Buhrow, Sarah A.; Reid, Joel M.; Ames, Matthew M.; Munn, David H.

CORPORATE SOURCE: Developmental Therapeutics Program, National Cancer Institute, Bethesda, MD, 20852, USA

SOURCE: Food and Chemical Toxicology (2008), 46(1), 203-211 CODEN: FCTOD7; ISSN: 0278-6915

PUBLISHER: Elsevier Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB 1-Methyl-D-tryptophan (D-1MT) reverses the immunosuppressive effect of indoleamine 2,3-dioxygenase (IDO), and it is currently being developed both as a vaccine adjuvant and as an immunotherapeutic agent for combination with chemotherapy. The present study examined the pharmacokinetics and toxicity of D-1MT in preparation for clin. trials. Incubation of D-1MT in rat blood plasma for 24 h produced no significant degradation, with <15% of D-1MT being bound to plasma protein. Following oral administration, D-1MT exhibited a larger AUC and Vd, longer elimination t1/2, and slower clearance in rats than in dogs. When oral doses of D-1MT exceeded levels of 600 mg/m2/day in rats, or 1200 mg/m2/day in dogs, the Cmax and AUC values decreased, resulting in a corresponding decrease in oral bioavailability. Thus, the doses were indicative of the lowest saturating doses in dogs and rats corresponding with an elimination t1/2 of 6.0 and 28.7 h, a Tmax of 1 and 8 h, and a bioavailability of 47 and 92%, resp. Tissue concns. of D-1MT in mice were highest in the kidney, followed by the liver, muscle, heart, lung, and spleen, resp.; 48 h post dosing, D-1MT was excreted in the urine (35.1%) and feces (13.5%). Oral administration of D-1MT in rats from 150 to 3000 mg/m2/day (25-500 mg/kg/day) and in dogs from 600 to 1200 mg/m2/day (30 and 60 mg/kg/day) for 28 consecutive days did not lead to mortality, adverse events, histopathol. lesions, or significant changes in hematol., clin. chemical, and body weight These results suggested that 3000 and 1200 mg/m2/day were the no-observed-adverse-effect levels in rats and dogs, resp. Mean plasma concns. of D-1MT (600 and 1200 mg/m2/day) in dogs 1 h post dosing were 54.4 and 69.5 μg/mL on Day 1, resp., and 53.1 and 66.6 μg/mL on Day 28, resp.; thus, indicating no increase in plasma D-1MT with a change in dose. In conclusion, D-1MT has little toxicity when administered orally to rats and dogs. Exceeding the saturating dose of D-1MT is unlikely to cause systemic toxicity, since any further increase in D-1MT plasma levels would be minimal.

REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 7 OF 13 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2007:843527 CAPLUS

DOCUMENT NUMBER: 147:400343

TITLE: Novel Tryptophan Catabolic Enzyme IDO2 Is the Preferred Biochemical Target of the Antitumor Indoleamine 2,3-Dioxygenase Inhibitory Compound D-1-Methyl-Tryptophan

AUTHOR(S): Metz, Richard; DuHadaway, James B.; Kamasani, Uma; Laury-Kleintop, Lisa; Muller, Alexander J.; Prendergast, George C.

CORPORATE SOURCE: Lankenau Institute for Medical Research, Wynnewood, PA, 19096, USA

SOURCE: Cancer Research (2007), 67(15), 7082-7087 CODEN: CNREA8; ISSN: 0008-5472

PUBLISHER: American Association for Cancer Research

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Small-mol. inhibitors of indoleamine 2,3-dioxygenase (IDO) are currently being translated to clinic for evaluation as cancer

therapeutics. One issue related to trials of the clin. lead inhibitor, D-1-methyl-tryptophan (D-1MT), concerns the extent of its biochem. specificity for IDO. Here, we report the discovery of a novel IDO-related Trp catabolic enzyme termed IDO2 that is preferentially inhibited by D-1MT. IDO2 is not as widely expressed as IDO but like its relative is also expressed in antigen-presenting dendritic cells where Trp catabolism drives immune tolerance. We identified 2 common genetic polymorphisms in the human gene encoding IDO2 that ablate its enzymic activity. Like IDO, IDO2 catabolizes Trp, triggers phosphorylation of the translation initiation factor eIF2α, and (reported here for the first time) mobilizes translation of LIP, an inhibitory isoform of the immune regulatory transcription factor NF-IL6. Tryptophan restoration switches off this signaling pathway when activated by IDO, but not IDO2, arguing that IDO2 has a distinct signaling role. Our findings have implications for understanding the evolution of tumoral immune tolerance and for interpreting preclin. and clin. responses to D-1MT or other IDO inhibitors being developed to treat cancer, chronic infection, and other diseases.

REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 8 OF 13 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2007:790312 CAPLUS

DOCUMENT NUMBER: 147:187318

TITLE: Indoleamine 2,3-dioxygenase inhibitor for enhancing immune response against tumor or infection and tryptophan metabolic product for suppressing immune response against transplant rejection and autoimmune disease

INVENTOR(S): Chen, Wei; Blazar, Bruce R.; Munn, David; Mellor, Andrew

PATENT ASSIGNEE(S): Medical College of Georgia Research Institute, Inc., USA

SOURCE: PCT Int. Appl., 93pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007081878	A2	20070719	WO 2007-US404	20070105
WO 2007081878	A3	20081224		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW			
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA			
EP 1981534	A2	20081022	EP 2007-717763	20070105
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, RS				

PRIORITY APPLN. INFO.: US 2006-756861P P 20060107
WO 2007-US404 W 20070105

AB The present invention provides methods for the control of the generation of regulatory T cells (Tregs) and uses thereof. Indoleamine 2,3-dioxygenase inhibitor e.g. 1-methyl-tryptophan is used to reduce immunosuppression mediated by regulatory T cells and to enhance immune response to vaccine, e.g. tumor or viral antigen. The invention also uses metabolic product of tryptophan for inducing regulatory T cells to increase immunosuppression and antigen tolerance to prevent and treat allograft or transplant rejection and autoimmune disease.

L3 ANSWER 9 OF 13 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2007:483054 CAPLUS
DOCUMENT NUMBER: 146:475678
TITLE: Indoleamine 2,3-dioxygenase modulation by TLR ligands and immunomodulatory uses thereof
INVENTOR(S): Mellor, Andrew; Munn, David
PATENT ASSIGNEE(S): Medical College of Georgia Research Institute, Inc., USA
SOURCE: PCT Int. Appl., 46pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007050405	A2	20070503	WO 2006-US40796	20061020
WO 2007050405	A3	20090423		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA				
AU 2006306521	A1	20070503	AU 2006-306521	20061020
CA 2626547	A1	20070503	CA 2006-2626547	20061020
EP 1937303	A2	20080702	EP 2006-836384	20061020
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, RS				
PRIORITY APPLN. INFO.:			US 2005-729041P	P 20051021
			WO 2006-US40796	W 20061020

AB The induction of indoleamine 2,3-dioxygenase (IDO) in an IDO-competent subset of dendritic cells by TLR ligands, including TLR9 ligands, and various uses thereof, are presented. Also presented are e.g. a method for enhancing an immune response by administration of a TLR9 agonist and an IDO inhibitor.

L3 ANSWER 10 OF 13 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2007:60697 CAPLUS
DOCUMENT NUMBER: 146:243247
TITLE: Inhibition of Indoleamine 2,3-Dioxygenase in Dendritic Cells by Stereoisomers of 1-Methyl-Tryptophan Correlates with Antitumor Responses
AUTHOR(S): Hou, De-Yan; Muller, Alexander J.; Sharma, Madhav D.; DuHadaway, James; Banerjee, Tinku; Johnson, Maribeth;

Mellor, Andrew L.; Prendergast, George C.; Munn, David H.
CORPORATE SOURCE: Immunotherapy Center and Departments of Pediatrics, Medicine, and Biostatistics, Medical College of Georgia, Augusta, GA, USA
SOURCE: Cancer Research (2007), 67(2), 792-801
CODEN: CNREA8; ISSN: 0008-5472
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Indoleamine 2,3-dioxygenase (IDO) is an immunosuppressive enzyme that contributes to tolerance in a number of biol. settings. In cancer, IDO activity may help promote acquired tolerance to tumor antigens. The IDO inhibitor 1-methyl-tryptophan is being developed for clin. trials. However, 1-methyl-tryptophan exists in two stereoisomers with potentially different biol. properties, and it has been unclear which isomer might be preferable for initial development. In this study, we provide evidence that the D and L stereoisomers exhibit important cell type-specific variations in activity. The L isomer was the more potent inhibitor of IDO activity using the purified enzyme and in HeLa cell-based assays. However, the D isomer was significantly more effective in reversing the suppression of T cells created by IDO-expressing dendritic cells, using both human monocyte-derived dendritic cells and murine dendritic cells isolated directly from tumor-draining lymph nodes. In vivo, the D isomer was more efficacious as an anticancer agent in chemo-immunotherapy regimens using cyclophosphamide, paclitaxel, or gemcitabine, when tested in mouse models of transplantable melanoma and transplantable and autochthonous breast cancer. The D isomer of 1-methyl-tryptophan specifically targeted the IDO gene because the antitumor effect of D-1-methyl-tryptophan was completely lost in mice with a disruption of the IDO gene (IDO-knockout mice). Taken together, our findings support the suitability of D-1-methyl-tryptophan for human trials aiming to assess the utility of IDO inhibition to block host-mediated immunosuppression and enhance antitumor immunity in the setting of combined chemo-immunotherapy regimens.
REFERENCE COUNT: 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 11 OF 13 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2006:387945 CAPLUS
DOCUMENT NUMBER: 144:404390
TITLE: Indolamine-2,3-dioxygenase inhibitors for modulation of immune regulation
INVENTOR(S): Pohl, Joerg; Niemeyer, Ulf
PATENT ASSIGNEE(S): Germany
SOURCE: Ger. Offen., 5 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 102004050111	A1	20060427	DE 2004-102004050111	20041013
PRIORITY APPLN. INFO.:			DE 2004-102004050111	20041013

AB The invention discloses the therapeutic application of indolamine-2,3-dioxygenase (IDO) inhibitors for the treatment of diseases related to untimely IDO gene expression.

L3 ANSWER 12 OF 13 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2004:1019533 CAPLUS
DOCUMENT NUMBER: 141:420433
TITLE: Use of inhibitors of indoleamine-2,3-dioxygenase in combination with other therapeutic modalities in the treatment of cancer and infection
INVENTOR(S): Munn, David; Mellor, Andrew
PATENT ASSIGNEE(S): Medical College of Georgia Research Institute, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 42 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 20040234623	A1	20041125	US 2004-780797		20040217
US 20050186289	A1	20050825	US 2004-780150		20040217
US 20090081155	A1	20090326	US 2008-175538		20080718
US 20090123420	A1	20090514	US 2008-175518		20080718
PRIORITY APPLN. INFO.:			US 2003-459489P	P	20030401
			US 2004-538647P	P	20040122
			US 2004-780150	A1	20040217
			US 2004-780797	A1	20040217

AB The invention discloses a method for treating a subject with a cancer or an infection, the method including administering an inhibitor of indoleamine-2,3-dioxygenase (IDO) in an amount effective to reverse IDO-mediated immunosuppression, and administering at least one addnl. therapeutic agent, wherein the administration of the inhibitor of IDO and the at least one addnl. therapeutic agent demonstrate therapeutic synergy.

L3 ANSWER 13 OF 13 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2003:818069 CAPLUS
DOCUMENT NUMBER: 139:322295
TITLE: Antigen-presenting cell populations and their use as reagents for enhancing or reducing immune tolerance
INVENTOR(S): Mellor, Andrew L.; Munn, David H.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 36 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20030194803	A1	20031016	US 2002-121909	20020412
CA 2483451	A1	20031023	CA 2002-2483451	20020412
WO 2003087347	A1	20031023	WO 2002-US11319	20020412

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

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     AU 2002307243        A1    20031027    AU 2002-307243         20020412
     AU 2002307243        B2    20080103
     EP 1501918           A1    20050202    EP 2002-807233         20020412
          R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
              IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
     US 20060292618       A1    20061228    US 2006-474162         20060623
     US 20070048769       A1    20070301    US 2006-474144         20060623
     AU 2008200315        A1    20080214    AU 2008-200315         20080122
PRIORITY APPLN. INFO.:                       AU 2002-307243      A3 20020412
                                             US 2002-121909      A  20020412
                                             WO 2002-US11319     W  20020412
AB   The disclosed invention is based on the discovery that antigen-presenting
     cells (APCs) may be generated to have predetd. levels of expression of the
     intracellular enzyme, indoleamine 2,3-dioxygenase (IDO).  Because
     expression of high levels of IDO is correlated with a reduced ability to
     stimulate T cell responses and an enhanced ability to induce immunol.
     tolerance, APCs having high levels of IDO may be used to increase
     tolerance in the immune system, as for example in transplant therapy or
     treatment of autoimmune disorders.  For example, APCs having high levels
     of IDO, and expressing or loaded with at least one antigen from a donor
     tissue may be used to increase tolerance of the recipient to the donor's
     tissue.  Alternatively, APCs having reduced levels of IDO expression and
     expressing or loaded with at least one antigen from a cancer or
     infectious pathogen may be used as vaccines to promote T cell responses
     and increase immunity.


=> d his

     (FILE 'HOME' ENTERED AT 09:56:45 ON 29 MAY 2009)

     FILE 'REGISTRY' ENTERED AT 09:56:57 ON 29 MAY 2009
L1             1 S 110117-83-4/RN

     FILE 'CAPLUS' ENTERED AT 09:57:32 ON 29 MAY 2009
L2            35 S L1
L3            13 S L2 AND (?CANCER? OR ?TUMOR? OR ?TUMOUR? OR ?NEOPLASM?)

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---Logging off of STN---


=>
Executing the logoff script...


=> LOG Y

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
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FULL ESTIMATED COST                                    49.96      52.71

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
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CA SUBSCRIBER PRICE                                   -10.66     -10.66

STN INTERNATIONAL LOGOFF AT 10:00:04 ON 29 MAY 2009
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